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SECOND SERIES VOL. 11, No. 11

BULLETIN

OF

THE NEW YORK ACADEMY OF MEDICINE

INCORPORATED 1851

NOVEMBER, 1935

PUBLISHED MONTHLY BY

THE NEW YORK ACADEMY OF MEDICINE

2 East 103 Street

New York, N. Y.

Invitation

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Calcium alone is insufficient!

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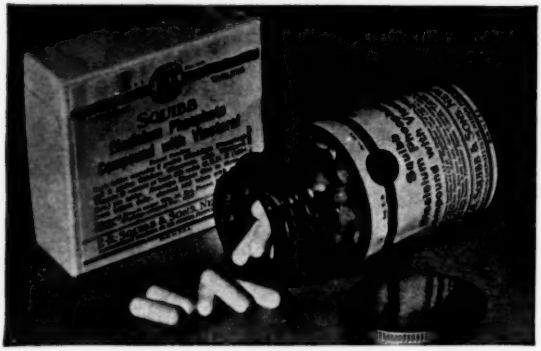
for normal growth and maintenance of bone structure



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EIGHTH ANNUAL GRADUATE FORTNIGHT

"Diseases of the Respiratory Tract"
October 21 to November 2, 1935

The Wesley M. Carpenter Lecture

ATELECTASIS, MASSIVE COLLAPSE AND RELATED POST-OPERATIVE CONDITIONS*

YANDELL HENDERSON
Professor of Applied Physiology
Yale University

In an address at a meeting of the American Medical Association twenty-four years ago, I pointed out that the methods of observation and of reasoning employed by physiologists in the study of normal processes should be applied to clinical problems also[1]. In other words, we should develop a clinical physiology. It is as a problem in clinical physiology that I shall here treat the subject of atelectasis, massive collapse, and related post-operative conditions.

The lungs are peculiar organs in that they have little independent activity or self regulation. Their activity is mainly determined and controlled by influences outside the lungs themselves. A muscle can be taken out of the body and made to contract. The heart can be excised and kept beating. The kidneys likewise, if perfused, will function independently of the rest of the body. But the lungs, once out of the chest and separated from the heart, are mere masses of tissue devoid of activity.

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* Delivered October 21, 1935. This is the only lecture of this year's Graduate Fortnight to be published by the Academy. All the papers presented at the evening meetings will be published in book form by W. B. Saunders Company.

If this is true of the normal physiology of the lungs, we should expect—and in fact we find—that in their abnormal physiology the dominant influences are generally not developed in the lungs themselves; they are induced by conditions in other parts of the body.

Massive collapse of an entire lung is so striking a phenomenon that some investigators have been tempted to look for its cause in an active contraction under some nervous influence. Some such reaction as this can be seen in a frog when the vagus is stimulated and the lung is thereby caused to contract. The human lung is permeated with non-striated muscle fibres[2] and it is probable that either over-activity or failure of these minute muscles plays an important part in asthma and emphysema. In general the elasticity of the various parts of the body is due rather to the muscular than to the connective tissues. We have learned recently that the elasticity of the systemic arterial system is partly due to the non-striated muscle fibres in the walls of the blood vessels[3]; and doubtless the muscular element in the structure of the lungs plays a large part in their elasticity. From their structure we should almost expect the lungs to exhibit contractions like the stomach or the bladder. But such a reaction, if it occurs at all in the development of atelectasis, is of relatively small importance compared to the mechanical influences that are extrinsic to the lungs[4].

Of all the influences upon the lungs involved in the development of post-operative atelectasis and massive collapse, by far the most important is that of the size of the thorax: its expansion or contraction. The size of the thorax is determined by the contraction or relaxation of the thoracic muscles. The intercostals, scaleni and levators, which lift and spread the ribs, play a considerable part. But it is the diaphragm that by its contraction and relaxation is the main factor in variations in the size of the thorax; and these variations extend from maximal inflation of the lungs down to atelectatic deflation. When Sir James Barr[5] first recognized massive collapse of a lung as a sequel

of a surgical operation, he came very close to the true explanation of its underlying cause; he assigned it to "paralysis of the diaphragm" and treated it successfully with "a course of respiratory gymnastics." And if we change the word paralysis to "abolition of tonus" and extend this conception of loss of tonus to all the respiratory muscles, and indeed to all the muscles of the body, we have before us the condition that underlies not only atelectasis and massive collapse, but also, as I shall try to show, practically all of the other features of post-operative depression of functions[6].

The full practical significance of tonus has not yet, I believe, been realized either by physiologists or clinicians. It is the essential feature in the physical depression that follows alike after surgical operations, physical injuries, anesthesia and severe illness. Tonus is chiefly manifested by muscles, but it is induced in the muscles largely by the influence of the nervous system, as well as by the metabolism and respiration of the muscles themselves. Tonus is thus an index of the state of the nervous system, and an expression of the vitality of the body as a whole.

Physiologists, especially those interested in the nervous system, understand fully that posture, particularly the ability to stand erect, or to hold up one's head, is dependent upon tonus. But the step from this physiological conception to its clinical corollary is not generally made. It is not sufficiently emphasized, or even generally realized, that when a patient is too weak to stand or even to hold up his head, his condition is generally one of extremely low tonus.

The significance of tonus in respiration[7], and particularly in the maintenance of the capacity of the thorax, may be illustrated by the following observations[8]: Some twenty years ago, while testing the efficiency of various methods of manual artificial respiration, I performed the following experiment. A cat was placed under moderate chloroform anesthesia; its trachea was connected with a small spirometer and the volume of air breathed in a minute was

recorded. A fatal dose of chloroform was then quickly administered and respiration, and the heart action, stopped finally. When the trachea was again connected with the spirometer and artificial respiration was performed by squeezing the thorax, a nearly normal volume of air was at first expelled from the lungs; and when the pressure upon the thorax was released the same volume of air was drawn in again. This inspiration was entirely due to the elastic recoil of the thoracic muscles and diaphragm. But, as the centers in the nervous system failed under asphyxia, the elasticity of the thorax was gradually abolished, and less and less air was drawn in each time that compression was released, until after about ten minutes, when the body was entirely flaccid, there was no movement of air whatever in or out of the lungs. Compression of the thorax by the operator's hands caused no air to be expired, for the chest was already deflated. And no air was drawn in, for the elastic recoil of the muscles was abolished with the entire loss of tonus.

This loss of tonus, abolition of elasticity and collapse of the chest is not a mere laboratory observation on animals. My associate, Dr. H. W. Haggard[9], has had an opportunity to make a similar observation on a man just dead from a heart attack. At first manual artificial respiration expelled air from the lungs and the tonic elasticity of the chest drew air in again. Then as the nerve centers controlling tonus failed under asphyxia, the elastic recoil of the respiratory muscles diminished until the thorax was deflated down to the size natural in a corpse.

A similar, if less extreme, observation was made twenty years ago in my laboratory upon normal men, that is medical students, as part of a regular class exercise. The manual prone pressure method of artificial respiration was applied by one student to another; and the volume of the pulmonary ventilation thus induced was determined. The volume of air exhaled and inhaled under this rhythmic compression of the chest was found to be in all cases almost exactly the same as that of natural breathing. This observation was at first very surprising, until its explanation was found. It means merely that the respiratory center regulates the tonus of the thoracic muscles and diaphragm to such a degree of elasticity that the chest expands exactly

enough alike after each normal expiration and each artificial compression to afford precisely the amount of pulmonary ventilation that the man normally needs to supply oxygen and remove carbon dioxide. The subjects were then directed to perform voluntary overbreathing for a couple of minutes, so as to overventilate the lungs. Because of the acapnia thus induced, the subject thereafter breathed subnormally for a time, or even stopped entirely. And when the rhythmic compression of the chest was applied during this period of apnea, it was found that much less air was moved out of the chest, and correspondingly little was drawn in.

More recently x-ray pictures have shown that in men during apnea, after forced breathing, the position of the chest is essentially that of passive expiration[10]. Evidently the elasticity that normally keeps the thorax expanded is not a mere mechanical condition, but rather a vital property under the control of the nervous system and particularly of that part of the nervous system that we call the respiratory center. If tonus is diminshed artificially by overbreathing, the elasticity is correspondingly diminished. In the depression of vitality that follows long anesthesia and extensive surgical operations, the chest cavity is so much decreased that the lungs are nearly deflated. A healthy man under normal conditions has more than twice as much air in his lungs as a corpse. It is probable that from the first breath of life until after the last the diaphragm is never completely relaxed.

Turning now to observations upon patients after surgical operations, it has been found by a number of observers[11] that the vital capacity, that is, the maximum volume of air that a patient can exhale may be decreased by as much as fifty per cent. In this decrease it is evident that the relaxation of the diaphragm plays a critical part, for it may be relaxed to such an extent that in x-ray pictures its shadow is as much as 7 or 8 centimeters headward from its normal position. This is as far or farther than the diaphragm moves under the deepest and most forcible voluntary expiration.

The sum and substance of all these observations is that when vitality is reduced the tonus of all the muscles in the body is correspondingly diminished. In the thorax par-

ticularly this decrease of tonus results in such a diminution of the size of the thoracic cavity and such a deflation of the lungs that many minute deaerated areas are produced.

This condition in its simple uncomplicated form has been well termed "collapse without symptoms[12]." When it is looked for it has been found after more than 80 per cent of all laparotomies. It is always accompanied by shallow respiration and rapid pulse.

This condition is so common that we may almost call it a normal sequel of major surgical operations. Why then, is massive collapse not more common than it is? Why does it occur in some cases and not in all? The reason for this is apparently one of pure chance. With the lungs partially deflated, it sometimes happens that mucus accumulates in one or another of the major airways until it is completely closed. In that part of the lung which is thus occluded, another development then sets in. The gases contained in the occluded lobe or lung are absorbed into the blood and the lobe or lung in the course of time—generally a rather short time—is completely collapsed. As one lung collapses it draws the mediastinum after it and atmospheric pressure inflates the other lung. Hence massive collapse is almost necessarily confined to one lung for the collapse of one lung tends to expand the other. We owe this conception to Dr. P. N. Coryllos[13].

The absorption of air from an occluded lung is not a vital reaction, but a purely physical process. It is distinct from the simple atelectasis from loss of tonus that we have been discussing. The preceding partial deflation permits the plugging of an airway. The closure of an airway, however produced, is then the sole causative condition. The air from an occluded lobe or lung is absorbed in exactly the same way that air is absorbed from any other body cavity such as the pleural cavity, into which air, or any other non-irritant gas, has been injected. The explanation of how and why a lung is collapsed by absorption of its gaseous contents, whenever the airway to that lung or lobe is blocked, is exactly the same as the explanation of the re-

absorption of the air from a pleural space and the consequent reinflation of a tuberculous lung that has been collapsed by pneumothorax. In both cases the absorption is due to the partial pressures of the gases concerned; and in neither case does blood pressure play any part whatever.

In brief, the physics of the process of absorption is as follows: In the lungs, so long as they are in free connection with the outside air, the total pressure of all the gases, oxygen, carbon dioxide, nitrogen, and water vapor, is the same as that of the atmospheric pressure in the outside air. Very nearly the same gas pressures prevail in the arterial blood as it leaves the lungs. In the tissues oxygen is absorbed and carbon dioxide is produced in nearly equal amounts. But owing to the difference in the manner in which oxygen and carbon dioxide respectively are held in the blood, the pressure of oxygen in the venous blood is greatly decreased, while the pressure of carbon dioxide is only slightly increased. The sum of the pressures of these two gases in the venous blood is therefore very much less than the sum of their pressures in the arterial blood. The partial pressure of nitrogen is, however, the same in both arterial and venous blood.

Suppose now that a few hundred cubic centimeters of air are injected into any body space, such for instance as the abdomen. At first carbon dioxide is given off from the blood and the tissues to the occluded air and part of its oxygen is absorbed. But the gaseous equilibrium that is thus approached, although never reached, approximates the gas pressure in the venous blood rather than the arterial blood; and more oxygen is therefore absorbed from this air than the carbon dioxide that diffuses into it. The volume of the air is thus decreased and as atmospheric pressure bears upon the exterior of the abdomen and as the soft tissues yield easily to atmospheric pressure, the space is decreased. As a result the nitrogen of the occluded air is now contained in a smaller space than before and its partial pressure, therefore, is raised above its original pressure and thus also above the pressure of nitrogen in the blood.

It is one of the fundamental laws of gases that each gas diffuses according to its own partial pressure regardless of what other gases may be present. Accordingly, a part of the nitrogen in this space that we are considering diffuses into the blood; and the total volume of the occluded gases is thus further decreased, and brings the partial pressure of the oxygen and carbon dioxide in the diminishing space again into relative excess above the pressures of these gases in the venous blood into which they therefore diffuse further. And, as a result of the continual interaction of these processes, any air that is injected into the abdominal cavity, or the pleural cavity, or any other body cavity is gradually absorbed under the influence of purely physical principles.

Similarly air that is occluded in a lung by a plug in a bronchus is gradually absorbed into the blood. And as it is absorbed, the lung is gradually collapsed under the pressure of the atmosphere bearing down through the trachea and bronchi into the other lung, and by elevation of the diaphragm by the pressure of the atmosphere upon the abdomen. This appears to be the complete explanation of why it is that a plug in a main bronchus results in collapse of the lung on that side, and an expansion of the other lung to fill part of the space.

The final confirmation of this purely physical explanation is that when, by means of bronchoscopy, a plug of mucus or other material is removed from a bronchus, the collapsed lung, as Chevalier Jackson[15] has shown, is rapidly reinflated by the negative pressure of the thorax aided by the movements of respiration. And, of course, the deeper the respiration, the greater the negative pressure and the greater the expansive force tending to reinflate the lung.

All of these facts point clearly to the conclusion that the decrease of vitality consequent upon major surgical operations involves a general decrease of body tonus. Owing to the decrease of tonus of the respiratory muscles and particularly of the diaphragm, the volume of the lungs is decreased to the point at which lobular atelectasis, collection of mucus, plugging of airways, and finally massive collapse may occur.

I shall not stop now to consider those cases, fortunately rather rare, in which infection develops in the occluded lung. I must, however, emphasize as strongly as I can, the point that the lungs are normally the best drained organs in the entire body, and that the airways are normally kept clear by ciliary movements and by the cough reflex: one of the most vigorous reactions of all those provided by Nature for the protection of the body. For this reason we should expect, as in fact we find, that occlusion of the airways plays an exceedingly serious and damaging part in pulmonary infections[16]. Pneumonia, as the internist sees it, is a topic outside the range of this

lecture, but I venture again[17] to point out, that in regard to pneumonia internists would do well to borrow from surgery one of its major axioms: a well-drained infection is relatively harmless; an occluded infection is always dangerous. Evidence is accumulating that those measures which surgeons and anesthetists find effective in preventing or relieving post-operative atelectasis and massive collapse are also useful if administered within 48 hours of the onset of pneumonia[18].

The gist of these measures, for the internist as for the surgeon, is as follows: Pure carbon dioxide is administered by means of an open mask in which the gas (2 to 3 liters a minute) mixes with the inhaled air. The inhalation is given for only a few minutes, but long enough and strong enough to induce marked hyperpnea. After five minutes rest it is repeated; and thereafter these two inhalations are administered at regular intervals of three to four hours. This technique is now extensively applied in the early stages of pneumonia by Dr. Lewis Gunther[19] and his colleagues in Los Angeles. He writes me that "the first inhalation generally causes severe coughing, which brings up surprisingly large amounts of yellow pus. The patient is considerably fatigued, but drops asleep, and awakens feeling and appearing better. Resolution occurs earlier than in untreated cases." Such observations, even though the series is still small, justify a wide trial, particularly in the early stages of pneumonia in children.

As the pulmonary circulation influences, and is influenced by, all of the conditions discussed in this paper, a few words on this subject are here in order: The volume of blood in the lungs and the volume flowing through the lungs may vary together or independently of each other. They are affected by three chief influences: (a) vasomotor nerves, (b) the efficiency of the heart, and (c) the degree of inflation of the lungs.

(a) Experimental evidence indicates that there are vasomotor nerves to the pulmonary blood vessels, and that the lungs form one of the reservoirs from which blood may be shifted when needed elsewhere. But it appears that this adjustment does not alter appreciably the volume of blood flowing through the lungs; for any restriction there would correspondingly diminish the circulation of the entire body: an effect like that of a pulmonary embolus.

(b) The left ventricle of the heart is both normally and abnormally the main factor determining the volume of blood in the lungs. Normally the diastolic receptive relaxation of that ventricle takes from the pulmonary veins in a minute exactly as much blood as the right heart pumps into the pulmonary artery; but never more than that volume. Such a condition as depletion of the pulmonary circulation is therefore impossible.[20] It never occurs. On the other hand, whenever the left ventricle fails to accept easily the volume of blood coming to it—whether because of valvular defects or impeded diastolic relaxation and filling—the volume of blood in the lungs is increased. The volume of blood flowing through the lungs is, however, determined almost entirely by the right heart and by the volume of the venous return to that side of the heart.

(c) Expansion of the thorax causes the lungs to hold, not only more air, but probably more blood also. When a lung or a part of a lung is collapsed, both the volume of blood in it and even more the volume flowing through it are decreased. The total flow through the lungs, however, is not decreased; instead, there is a compensatory increase of flow through the lung, or parts of the lung, that are still inflated; and the blood is thus aerated. This adjustment is seen in patients in whom a therapeutic pneumothorax has been produced. A tuberculous patient is not rendered cyanotic when one lung is partially, or even completely, deflated.

I return now to post-operative depression and particularly to the depression of the circulation that often accompanies atelectasis. This is the condition which, in its extreme form, was formerly called "surgical shock." I say formerly, because I find that no self-respecting surgeon is now willing to admit that any of his patients ever reaches a condition that he is willing to have a mere physiologist call "shock." I will, therefore, speak of this condition only as "post-operative circulatory depression."

The large majority of all human beings die through gradual depression and final failure of the circulation[21]. As the result of a vast amount of investigation we know now what factor in the circulation it is that fails; but until recently we have not known just how or why this factor fails. Here normal physiology and clinical physiology present two closely related problems; one the converse of the other. One is the problem of how in health the normal circulation is maintained; the other is the problem of how in illness and vital depression the circulation fails. We know that the heart is a pump that, so to speak, lifts the blood from the

low pressure in the veins up to the high pressure in the arteries and we know that the vasomotor nervous system controls the distribution of the blood and maintains arterial pressure. But the volume of blood that the heart can pump is limited by the volume that comes to it through the veins from the reservoirs of blood in the tissues. Thus beside the heart and the vasomotor mechanism, there is a third major factor in the circulation: the factor that determines the volume of blood returning to the heart through the veins. To this factor I gave the name of the "venopressor mechanism" and it is now generally recognized that in the great majority of cases depression of the circulation is not due to failure of the heart, nor of the vasomotor system. It is due rather to a gradual decrease in the volume of blood returning to the heart: that is, to the progressive depression and final failure of the venopressor mechanism.

Recently we have made observations in New Haven from which we can define the nature of the venopressor mechanism; and we find that the principal factor in it is the general tonus of the musculature of the body, both skeletal and visceral[22]. Everyone recognizes that in the physical depression following a major surgical operation, or after a serious illness or an attack of influenza, there are two outstanding conditions: the tonus of the body is lessened and the circulation is weakened. In addition, in post-operative depression without infection or fever, there is also a decrease of metabolism, oxygen consumption and heat production. Heretofore, the decrease of tonus has generally been imputed to a poor circulation. In reality, as facts I am about to state seem to me to indicate, the poor circulation is due to a diminished venous return to the heart, which in turn along with the depression of metabolism is due to lowered body tonus.

It is well recognized that every movement of the limbs and every contraction of the abdominal muscles tends to promote the flow of blood through the venous system back to the heart. But it has not heretofore been realized that in the healthy body, the tonic contraction of the muscles main-

tains continually a pressure within the tissues appreciably above that of the outside atmosphere. This pressure between the fibres of a muscle under tension arises in much the same way that pressure between the strands of a rope is produced by a longitudinal pull upon the rope. The pressure amounts, even in a relaxed but still tonic muscle, to 50-70 mm. of water column during complete rest. When muscle tonus is increased, the pressure is higher, up to 90 mm. or more. When tonus is abolished, as it is in a moribund man or animal, the internal pressure is also abolished. The method of measuring this pressure is to thrust a hypodermic needle into the middle of a muscle, usually the biceps, and to determine the pressure required to induce a minute amount of saline to run in. This intramuscular pressure is too small to have any considerable direct effect upon arterial blood pressure and the flow of blood into a muscle; but it is quite sufficient to exert a strong influence upon the venous outflow from a muscle. It is one of the first principles of hydraulics that a fluid tends to flow from a place of high pressure to one of lower pressure; and the greater the difference of pressure the more rapid the flow.

I am not for a moment denying or questioning the correctness of the opinion that the entire energy of the circulation during bodily rest is due to the push from behind imparted to the blood by the left heart. But, in order to push the blood not only from the heart to the tissues, but also onward from the tissues back through the veins to the heart, it is essential that some of the force imparted by the heart shall be conserved up to the completion of the circuit. And so long as the tissues of the body have tonus, elasticity and an internal pressure, a part of the force imparted to the blood by the heart is retained by the blood in the vessels within and beneath the muscles. It is this force that presses the blood on into the veins, and thus supplies the right heart and supports the circulation. On the other hand, when muscle tonus is depressed or fails, the entire force of arterial pressure is lost in the flaccid tissues and the blood stagnates there instead of flowing on back to the right heart.

Nor is this all. As we have seen, the depression of muscular tonus permits the thorax to diminish in size and the lungs to collapse toward atelectasis. And thereby another handicap is imposed upon the circulation; for as the lungs deflate, the negative pressure of the intrapleural space decreases. So long as the thorax and lungs are normally expanded this negative pressure is one of the main accessory factors aiding in drawing blood into the right heart. When the tonus of the body is diminished, not only is the positive pressure in the body as a whole diminished correspondingly, but the negative pressure of the thorax also.

The point to be emphasized is that the part played by general body tonus is distinct from that of vasomotor tonus. Vasomotor tonus determines the height of arterial pressure. The general tonus of the body and intra-tissue pressure determine the volume of the venous return to the heart and the volume of the output of the heart. In progressive vital depression it is general tonus that fails first. The output of the heart is thus diminished. It is only after the volume of the circulation has been thus greatly decreased that arterial pressure fails. Almost to the last the vasomotor mechanism, instead of failing, strives by an extreme constriction of the arteries and arterioles to maintain arterial pressure in spite of the diminishing output of the heart. This is the process leading to the large majority of all deaths.

Evidently the general tonus of the body is a function of supreme importance. When we learn how to conserve it, we shall at the same time have learned both how to prevent atelectasis and also how to prevent depression and failure of the circulation: in other words, to prevent patients from dying. And undoubtedly, without fully realizing it, internists dealing with prostrating illness and surgeons and anesthetists dealing with serious operations have learned to avoid those procedures that tend to diminish tonus and to utilize those that tend to conserve this supremely important factor in vitality. But the explicit recognition that it is tonus that must be conserved, will be a long step forward. We shall understand much that is now obscure.

As an illustration take the subject of spinal anesthesia. Atelectasis follows spinal anesthesia almost or quite as frequently as it does inhalational anesthesia. And this is exactly what we should expect when we realize that spinal anesthesia not only blocks off sensation, it also abolishes tonus in those parts of the body that are anesthetized and probably in many cases it also depresses the tonus of the entire body.

Another feature of spinal anesthesia is that even a quite moderate loss of blood is sometimes fatal, as should be expected, when tonus has been depressed and a large part of all the blood in the body rendered more or less stagnant.

A third feature of spinal anesthesia is highly significant. Pulmonary complications are fairly common sequelae of spinal anesthesia, yet no direct irritation of the lungs possibly occurs. It is logical, therefore, to infer that when atelectasis follows an inhalational anesthesia the cause is not direct pulmonary irritation, but diminished bodily tonus. Irritation of the lungs by the gases of war induces edema, not massive collapse[23]. There is therefore, little if any ground left for the belief once general that irritation of the lungs by the vapor of ether or other volatile anesthetics is the cause of pulmonary complications after inhalational anesthesia. The underlying cause of post-operative atelectasis after any form of anesthesia, alike spinal or inhalational, is diminished body tonus and the consequent deflation and collapse of the lungs.

Finally we face the questions: Normally, what are the factors that cause and maintain tonus? Abnormally, why does tonus fail?

In the main, certainly, the tonic contractions of striated muscles are induced through the same nerve fibres through which voluntary contractions are mediated. Many influences bear upon the anterior horn cells in the spinal cord from which the motor fibres come. Among these influences the proprioceptor afferent fibres from the muscles themselves play an important part in the tonus that maintains

the erect position. Both the cerebrum and the cerebellum exert powerful influences upon the tonic nerve impulses discharged from the motor centers in the spinal cord.

There are also reflex influences from the skin. Even such mild stimulation of the skin as that afforded by a comfortably cool temperature and air movement in a well ventilated room has an influence in maintaining a good tonus. On the other hand, in a badly ventilated and uncomfortable room this influence results in a decrease of tonus and induces a diminished intramuscular pressure[24].

The respiratory center exerts at times a dominating control, not only over the respiratory muscles, but over the entire musculature of the body. Even the slight depression of respiration induced by voluntary overbreathing causes a marked decrease in the tonus of all of the respiratory muscles. Profound anesthesia may stop respiration and abolish tonus. The demand of the surgeon for extreme relaxation increases the tendency to serious post-operative sequelae. On the other hand, stimulation of respiration by carbon dioxide increases not only the volume of breathing, but also the tonus of the respiratory and other muscles. The thorax is expanded, and the lungs are cleared[25]. The venous return to the right heart is increased; and the circulation is thus restored and supported.

There remains however much still to be learned regarding the normal physiology of tonus, and far more regarding the clinical physiology of depression of tonus.

When that fuller knowledge is gained it will doubtless yield means and methods of resuscitation more effective than any now available. But even then, and always, it will be necessary to keep in mind that every measure of restoration may fail, if atelectasis has gone too far; if a plug is too firmly impacted in a bronchus; or if the circulation has failed to the point of asphyxia of the tissues. Far more important than any possible measure of restoration is the conservation of the patient's vitality. The requirements for

conservation are skillful anesthesia and surgery that traumatizes as little as possible. The extent to which vitality has been depressed or conserved is shown in the patient's tonus after the operation. To a very large extent vitality is tonus.

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Library Notes

FIELDING H. GARRISON

Articles and Book Reviews in the *Bulletin of The New York Academy of Medicine*

At the suggestion of Mr. Leonard L. Mackall, one of our Library Consultants, this list of the late Col. Fielding H. Garrison's articles and book reviews published in the *Bulletin of The New York Academy of Medicine,* has been compiled by members of the staff of the Library. The entries are numbered and arranged alphabetically under subject; but in addition, cross references to the reviews are given under the names of authors. In the case of papers which have two or more subjects, cross references from these are included. For the sake of those who keep their *Bulletin* unbound, Mr. Mackall suggested that in the references the month be given. Col. Garrison's articles were much appreciated when they appeared, and it is hoped that this subject list will prove helpful.

ABBOTT, M., *see* no. 55.

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SINGER, C., *see* no. 3.

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Hague, Nijhoff, 1935, v. 1.

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Weiss, S. Diseases of the liver, gallbladder, ducts & pancreas, their diagnosis & treatment.
N. Y., Hoeber, 1935, 1099 p.

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London, Churchill, 1935, 543 p.

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London, Lane, [1935], 215 p.

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London, Arnold, 1935, 492 p.

October

Abramson, H. A. Electrokinetic phenomena and their application to biology and medicine.
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Andrews, (Mrs.) M. R. (Shipman). A lost commander: Florence Nightingale.
Garden City, Doubleday, 1935, 299 p.

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Beck, A. C. Obstetrical practice.
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Beer, E. Tumors of the urinary bladder.
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Beesly, L. & Johnston, T. B. A manual of surgical anatomy. 4. ed.
N. Y., Oxford Univ. Press, [1935], 717 p.

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London, Milford, 1935, 382 p.

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N. Y., Harper, 1935, 346 p.

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Phil., Lippincott, [1935], 5v.

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Franklin, E. C. The nitrogen system of compounds.
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Heaton, C. E. Modern motherhood.
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Huntley, K. E. Financial trends in organized work in New York City.
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Johnston, T. B. A synopsis of regional anatomy. 3. ed.
Phil., Lea, 1935, 460 p.

Joslin, E. P. The treatment of diabetes mellitus. 5. ed.
Phil., Lea, 1935, 620 p.

Kennedy, A. J.; Farra, K. [et al.]. Social settlements in New York City.
[N. Y.], Welfare Council, 1935, 599 p.

Kessler, H. H. The crippled and the disabled.
N. Y., Columbia Univ. Press, 1935, 337 p.

Knowles, F. C. Diseases of the skin. 3. ed.
Phil., Lea, 1935, 640 p.

Kovács, R. Electrotherapy and light therapy. 2. ed.
Phil., Lea, 1935, 696 p.

Lukis, D. H. Problems of anaesthesia in general practice.
London, Hodder, 1935, 158 p.

Marshall, C. S. An introduction to human anatomy.
Phil., Saunders, 1935, 385 p.

Novak, E. The woman asks the doctor.
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Obrig, T. E. Modern ophthalmic lenses and optical glass.
N. Y., [Author], 1935, 323 p.

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N. Y., Vanguard Press, [1935], 297 p.

Pearson, W. J. & Wyllie, W. G. Recent advances in diseases of children. 3. ed.
London, Churchill, 1935, 566 p.

Perla, D. & Marmorston, J. The spleen and resistance.
Balt., Williams, 1935, 170 p.

Pickworth, F. A. Chronic nasal sinusitis and its relation to mental disorder.
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Reed, R. The illegitimate family in New York City.
[N. Y.], Welfare Council, 1934, 385 p.

Rettger, L. F.; Levy, M. N.; Weinstein, L. [et al.]. Lactobacillus acidophilus and its therapeutic application.
New Haven, Yale Univ. Press, 1935, 203 p.

Rice, T. B. A textbook of bacteriology.
Phil., Saunders, 1935, 551 p.

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Phil., Lea, 1935, 367 p.

Rubin, H. H. The glands of life.
N. Y., Bellaire, [1935], 164 p.

Schilder, P. The image and appearance of the human body.
London, Paul, 1935, 353 p.

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[Milano, Sormani], 1934, 2v.

Speed, K. A text-book of fractures and dislocations. 3. ed.
Phil., Lea, 1935, 1000 p.

Whiting, M. H. Ophthalmic nursing. 2. ed.
London, Churchill, 1935, 191 p.

Zilboorg, G. The medical man and the witch during the Renaissance.
Balt., Johns Hopkins Press, 1935, 215 p.

PROCEEDINGS OF ACADEMY MEETINGS

OCTOBER

STATED MEETINGS

SPECIAL NOTICE

There was no Stated Meeting of the Academy on October 3.

The next Stated Meeting (Harvey Society) was that of October 17.

THE HARVEY SOCIETY (IN AFFILIATION WITH THE NEW YORK ACADEMY OF MEDICINE)

October 17

THE FIRST HARVEY LECTURE, "The Proteins and Their Enzymic Degradation," Max Bergmann, Member of the Rockefeller Institute for Medical Research.

SECTION MEETINGS

SECTION OF DERMATOLOGY AND SYPHILOLOGY—October 1

I. READING OF THE MINUTES.

II. PRESENTATION OF MISCELLANEOUS CASES.

III. DISCUSSION OF SELECTED CASES.

IV. EXECUTIVE SESSION—Examination of cases is limited to members and their invited guests.

SECTION OF SURGERY—October 4

I. READING OF THE MINUTES.

II. PRESENTATION OF CASES—a. 1. A compound depressed fracture of the skull of a child with almost instantaneous onset of Jacksonian seizures; 2. The therapeutic injection of air for the relief of longstanding symptoms following skull fracture; a case with surprising result, Howard Patterson; b. Cases illustrating the papers of the evening, Byron Stookey, Edwin G. Ramsdell; Discussion by Ellsworth Eliot.

III. PAPERS OF THE EVENING—a. The diagnosis and management of spinal cord injuries, Byron Stookey; Discussion by C. R. Murray, J. E. J. King. b. Skull fractures, a study of 100 consecutive cases, Edwin G. Ramsdell; Discussion by Frederic W. Bancroft, S. Bernard Wortis, Foster Kennedy, C. Hirsch, C. R. Murray.

IV. GENERAL DISCUSSION.

V. EXECUTIVE SESSION.

SECTION OF NEUROLOGY AND PSYCHIATRY—October 8

I. PRESENTATION OF CASES—a. Metastatic melanoma involving the nervous system, Herman Wortis (by invitation), S. Bernard Wortis; Discussion, Foster Kennedy; b. A case of neuromyelitis optica, E. D. Friedman; Discussion, Israel Wechsler, Marcus Neustaedter, Foster Kennedy, S. B. Wortis, A. M. Rabiner.

II. PAPERS OF THE EVENING—a. The effect of experimental lesions of the superior cerebellar peduncle in macacus rhesus monkeys, Armando Ferraro, S. E. Barrera (by invitation); Discussion, Henry Alsop Riley, Kurt Goldstein; b. The treatment of general paralysis by combined electropyrexia and tryparsamide, L. E. Hinsie, J. R. Blalock (by invitation).

SECTION OF PEDIATRICS

The regular October meeting of the Section of Pediatrics was not held.

SECTION OF MEDICINE—October 15

I. READING OF THE MINUTES.

II. The present status of immunization in pertussis and poliomyelitis, William H. Park (45 minutes); Discussion by, Thomas E. Waldie (5 minutes), M. Brody (5 minutes) (by invitation).

III. Meningitis in man caused by a filterable virus, Thomas M. Rivers and T. P. McNair Scott (45 minutes) (by invitation); Discussion by, Harold G. Wolff (5 minutes) (by invitation).

SECTION OF GENITO-URINARY SURGERY—October 16

I. READING OF THE MINUTES.

II. PRESENTATION OF CASES—a. Diversion of the urinary stream (Folsom) in a case of carcinoma of the bladder, John W. Draper (by invitation); b. Successful surgical treatment of one case of bichloride anuria, William R. Delzell.

III. PAPER OF THE EVENING—a. Chemical nephrosis, Leon Herman (by invitation); Discussion, Alexander Gettler, Thomas A. Gonzales, Orrin S. Wightman, George F. Cahill, Lewis T. Mann, Edward C. Brenner, Irving Simons.

COMBINED MEETING OF THE SECTION OF OTOLARYNGOLOGY and THE NEW YORK ROENTGEN SOCIETY—October 16

PAPER OF THE EVENING—Roentgen therapy of epitheliomas of the tonsillar region, hypopharynx and larynx. (An extension of observations reported before the American Roentgen Ray Society in 1931.) Henri Coutard, Chief of the Department of Roentgen Therapy for Cancer, Radium Institute of the University of Paris (Curie Institute); Discussion by, Maurice Lenz, William Harris, James Ewing, Francis Carter Wood, Robert E. Buckley, Charles J. Imperatori, Henry B. Orton.

Due to the length of the program, it was not feasible to invite open discussion.

SECTION OF ORTHOPEDIC SURGERY—October 18

I. READING OF THE MINUTES.

II. PAPERS OF THE EVENING—a. Osteomyelitis, Abraham O. Wilensky; b. The time for operation in acute osteomyelitis, Paul Colonna; c. Bacteriophage in osteomyelitis, George L. Inge (by invitation); d. Rationale and results of maggot therapy in chronic osteomyelitis, Joseph Buchman.

SECTION OF OPHTHALMOLOGY—October 21

The Section decided to forego its October meeting in favor of the Graduate Fortnight. The program of the Graduate Fortnight for Monday evening, October 21, has been arranged by courtesy of the Section.

SECTION OF OBSTETRICS AND GYNECOLOGY—October 22

The Section decided to forego its October meeting in favor of the Graduate Fortnight. The program of the Graduate Fortnight for Tuesday evening, October 22, has been arranged by courtesy of the Section.

NEW YORK PATHOLOGICAL SOCIETY *in affiliation with* THE NEW YORK ACADEMY OF MEDICINE

There was no meeting of the New York Pathological Society in October, as the Graduate Fortnight of the Academy of Medicine was given during a period including the date of the regular meeting.

NEW YORK MEETING OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE

October 16

I. Metabolism of bromobenzene in growing dogs and mice maintained on an adequate diet, J. A. Stekol.

II. Effect of anterior hypophysis emulsion on natural resistance of hypophysectomized and normal rats to histamine poisoning, D. Perla.

III. Effect of Vitamin A on proliferation of fibroblasts, Lillian E. Baker (Introduced by T. M. Rivers).

IV. Influence of certain dye-stuffs on fermentation and respiration of yeast extract, L. Michaelis, C. V. Smythe.

V. Experiments on purification of bacteriophage, and a respiratory pigment in *Escherichia coli communis*, K. Meyer, R. Thompson, D. Khorazo, J. W. Palmer.

VI. Effectiveness of the Shope papilloma virus in various American rabbits, J. W. Beard, Peyton Rous.

VII. Certain factors determining the course of virus induced tumors, John G. Kidd, Peyton Rous, J. W. Beard.

VIII. Changes in preparation of type specific polysaccharides of pneumococcus necessitated by a quantitative study of precipitation in rabbit antisera, M. Heidelberger, F. E. Kendall, H. W. Scherp.

IX. Agglutination and precipitation reactions between hemolytic streptococci of Groups A through G (Lancefield) and sera of patients with rheumatoid arthritis, C. McEwen, H. Chasis, R. C. Alexander (Introduced by E. P. Ralli).

DEATHS OF FELLOWS OF THE ACADEMY

CAILLÉ, AUGUSTUS, Ph.G., M.D., 121 East 60th Street, New York City; graduated from the New York College of Pharmacy in 1873 and from the medical schools of the Universities of Wurzburg, Germany, 1877, and Columbia, 1881; elected a Fellow of the Academy, April 2, 1885; died October 10, 1935. Dr. Caillé was consulting physician at Post-Graduate Hospital and for thirty-five years, from 1888 to 1923, he was Professor of Medicine and Children's Diseases at the New York Post-Graduate Medical School and Hospital. He was Consulting Physician at Lenox Hill Hospital where he had been on the staff for fifty years. He was a member of the County and State Medical Societies, and a Fellow of the American College of Physicians and the American Medical Association. He was a member and a former president of the American Pediatric Society.

WHITE, JOHN BLAKE, M.D., 164 East 72nd Street, New York City; graduated from the College of Physicians and Surgeons in 1874; elected a Fellow of the Academy June 1, 1882; died November 4, 1935. Dr. White was a member of the County and State Medical Societies, the American Therapeutic Association, the American Public Health Association, and a Fellow of the American Medical Association. At the time of his death he was consulting physician at the Whitefield Hospital, Littleton, New Hampshire.

THE GRADUATE FORTNIGHT OF 1935

In January the Committee on Medical Education chose 'Diseases of the Respiratory Tract' as the general subject for the Eighth Annual Graduate Fortnight. A Subcommittee was appointed and the dates were set as October 21st to November 2nd. It was decided to present the same features as in previous years, that is, evening meetings to be held at the Academy, afternoon clinics to be held in selected hospitals (eighteen being invited to participate), and an exhibit. A director of the exhibit was appointed and a Committee to assist him. A Committee on Therapeutics was also appointed. A registration fee of $3.00 was decided upon.

Two speakers for each evening meeting were invited and were asked to limit their talks to about forty minutes each, in order that the meetings might be concluded by ten o'clock. Representatives of hospitals formed a Committee on Clinics and met several times with the Subcommittee to arrange and coordinate clinical programs.

In April the Council approved the publication of the papers to be presented at the evening meetings and W. B. Saunders Company was engaged to bring out the volume.

Publicity

A preliminary announcement, with return post card attached, was mailed to over twenty-one thousand registered physicians living within a radius of one hundred miles of New York City. Reading notices in the form of releases were sent to most of the important medical journals of the country. The complete program was printed in June and mailing was begun the latter part of the month. Three thousand four hundred and ninety-five requests were received for copies of the complete program.

Registration and Ticket Distribution

A total of six hundred and fifty-eight physicians registered for the Fortnight and were issued general admission tickets. These registrants came from twenty-six states and five foreign countries. About two-thirds of them came from New York City. Fellows and Members of the Academy were issued admission cards without charge.

Attendance at evening metiengs averaged about six hundred and fifty. At two of the meetings there was an attendance of over nine hundred. The smallest attendance was four hundred and twenty-three.

The afternoon clinics were very well attended.

The exhibit of this year was larger and appeared to be better attended than that of any previous Graduate Fortnight. To visit the exhibit no tickets were required.

THE GRADUATE FORTNIGHT OF 1935

REGISTRATION—TOTAL 658

From New York State—494

Manhattan	252	Bronx	77
Brooklyn	58	Queens	28
		Richmond	7

New York commuting area (25-mile radius), 17; Up-State New York, 55.

From New Jersey—84

Commuting area (25-mile radius), 74; other parts, 10.

From 31 Other States and Countries—80

Arkansas	1	North Carolina	1
California	2	Ohio	2
Connecticut	15	Pennsylvania	14
District of Columbia	1	Rhode Island	1
Florida	2	Tennessee	1
Illinois	4	Texas	1
Indiana	3	Virginia	4
Iowa	1	Washington	1
Kansas	1	West Virginia	1
Louisiana	2	Wisconsin	4
Maine	1		
Massachusetts	1	Puerto Rico	3
Michigan	1	Canada	4
Minnesota	1	Cuba	3
Mississippi	1	Belgium	1
Nebraska	1	South Australia	1

OFFICERS OF SECTIONS AND AFFILIATED SOCIETIES, 1935-36

Dermatology and Syphilology, 1st Tuesday

Chairman	*Secretary*
MAX SCHEER	FRANK C. COMBES, JR.
509 Madison Avenue	80 West 40 Street

Surgery, 1st Friday

CONDICT W. CUTLER, JR.	WILLIAM F. MACFEE
667 Madison Avenue	1107 Fifth Avenue

Neurology and Psychiatry, 2nd Tuesday

LEON H. CORNWALL	ABRAHAM A. BRILL
30 East 76 Street	1 West 70 Street

Historical and Cultural Medicine

2nd Wednesday of November, January, March and May

JEROME P. WEBSTER	REGINALD BURBANK
620 West 163 Street	6 East 78 Street

Pediatrics, 2nd Thursday

BELA SCHICK	ALEXANDER T. MARTIN
17 East 84 Street	107 East 85 Street

Ophthalmology, 3rd Monday

JOHN H. DUNNINGTON	LEGRAND H. HARDY
30 West 59 Street	30 East 40 Street

Medicine, 3rd Tuesday

PAUL REZNIKOFF	CLARENCE E. DE LA CHAPELLE
525 East 68 Street	140 East 54 Street

Genito-Urinary Surgery, 3rd Wednesday

JOSEPH A. HYAMS	ROY B. HENLINE
78 East 79 Street	901 Lexington Avenue

Otolaryngology, 3rd Wednesday

WESTLEY M. HUNT	CHARLES W. DEPPING
33 East 68 Street	19 West 54 Street

Orthopedic Surgery, 3rd Friday

WALKER E. SWIFT	NICHOLAS S. RANSOHOFF
115 East 61 Street	1100 Park Avenue

Obstetrics and Gynecology, 4th Tuesday

WALTER B. MOUNT	ARTHUR M. REICH
21 Plymouth St., Montclair, N. J.	141 West 77 Street

AFFILIATED SOCIETIES

New York Roentgen Society, 3rd Monday

President	*Secretary*
RAMSAY SPILLMAN	E. F. MERRILL
115 East 61 Street	30 West 59 Street

Society for Experimental Biology and Medicine, 3rd Wednesday

E. L. OPIE	A. J. GOLDFORB
1300 York Avenue	City College, Convent Ave. & 139 St.

Harvey Society, 3rd Thursday

R. KEITH CANNAN	RANDOLPH WEST
Bellevue Hospital	622 W. 168 Street

New York Pathological Society, 4th Thursday

WM. C. VON GLAHN	IRVING GRAEF
622 West 168 Street	Bellevue Hospital